an endothelial dependent vasodilator. Finally, 10 mM sodiumnitrite (an endothelial independent vasodilliator) was added.

The patient group consisted of 37 patients, with a mean age of 61.4 (± 8.4) years, 27% was female and the mean dilation to ME was 40% (± 24%) of the precontraction to PE. Linear regression showed that both total serum cholesterol (regression coefficient (r.c.) = 10.7%/mmol, p = 0.008) and ldl-cholesterol (r.c. = 11.5%/mmol, p = 0.01) were predictors for impaired endothelial dependent dilation. Adjusted for several other clinical characteristics in a multiple linear regression model, total serum cholesterol was the only statistically significant predictor of endothelial dysfunction (r.c 10.6%/mmol, p = 0.006). Endothelial independent vasodilation to sodiumnitrite was not influenced by serum lipid levels.

These results indicate that total cholesterol is the best predictor for endothelial dysfunction in IMA's of coronary bypass patients.

921-111 Plaque rupture in men is associated with high serum cholesterol

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The association between coronary plaque rupture and risk factors has not been fully explored. We prospectively examined 164 hearts from men dying unexpectedly; there were 44 sudden coronary deaths (SCD) with plaque rupture (age 48 ± 9), 53 SCD with stable plaque with or without healed myocardial infarction (age 53 ± 11), 16 SCD with eroded plaques (age 46 ± 9) and 51 non-coronary deaths (age 49 ± 10 years). Hearts were perfusion fixed at physiologic pressures and coronary arteries sectioned serially. All areas of cross sectional luminal narrowing ≥50% were evaluated histologically for thrombi. Postmortem sera were evaluated for % glycosylated hemoglobin, thiocyanate as an indicator for cigarette smoking, total cholesterol (TC), and high density lipoprotein cholesterol (HDL-C). Mean TC/HDL was 8.5 ± 0.6 in SCD with plaque rupture, exceeding controls (5.0 ± 0.3, $p < 0.0001$), SCD with stable plaque (5.5 ± 0.6, $p < 0.0001$), and SCD with eroded plaque (5.0 ± 1.9, p = 0.002). By logistic regression, TC/HDL-C was a predictor of plaque rupture independent of age, glycosylated hemoglobin, heart weight, smoking history, and hypertension (p = 0.0008, odds ratio 5.1). These data suggest that the reduced morbidity and mortality by cholesterol lowering in primary and secondary prevention is related to the stabilization of plaques with prevention of plaque rupture.

921-112 Soluble Cell Adhesion Molecules Are Regulated by Plasma Cholesterol in Familial Hypercholesterolemia

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How cholesterol (CH) lowering produces clinical benefits, besides the angiographic evidences, it is unknown. In our study on the effects of low density lipoproteins (LDL)-apheresis in patients with diet and drug resistant familial hypercholesterolemia (FH) we addressed the specific question if high plasma CH levels "per se" may adversely affect endothelium adhesiveness and if this phenomenon might be reversible.

We studied, in 8 FH patients, the acute and after 2 and 6 days effect of CH removal on plasma intercellular adesion molecule 1 (sICAM1) and on endothelium leukocyte adhesion molecule 1 (sELAM1). Apolipoprotein B containing lipoproteins were selectively absorbed on column of destran sulfate cellulose and during 3.5–4 hours a plasma volume of 6.5–9.2 litres was treated. CH, CH-LDL, apo B, TG and Lp(a), were reduced by 74%, 82%, 79%, 56%, 86%, respectively. No significant effect was observed on HDL-CH. Clinical chemical and biocompatibility showed minimal changes. Basal sICAM1 and sELAM1 levels were higher compared to healthy control subjects; after, and not by, ldl-apheresis they were constantly and significantly ($p < 0.0001$ and $p < 0.0004$, respectively) reduced. Individual, pre and post treatement, values of both sICAM1 and sELAM1 were positively and significantly ($p < 0.0001$ and $p < 0.02$, respectively) correlated with total CH. Rebound sICAM1 and sELAM1 curves showed a pattern similar to that of total CH but not TG and Lp(a).

In the absence of changes of tumor necrosis factora and factors involved in inflammation, these results indicate a possible role for CH in regulating endothelium adhesiveness at least in FH and confirm, in a clinical setting, the upregulation of endothelium adhesiveness observed in atherogenesis induced in animal rendered hypercholesterolemics.

921-113 Influence of Hyperlipidemia on Restenosis After Coronary Artery Stent Implantation

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Restenosis after percutanous coronary angioplasty (PTCA) depends on various mechanisms such as intimal hyperplasia, elastic recoil, arterial remodelling and smooth muscle proliferation, whereas restenosis after stent implantation is predominantly determined by neointimal proliferation. Hypercholesterolemia is known to be a major risk factor for coronary artery disease, but the association with neointimal proliferation has not been fully elucidated. Therefore, the role of lipids on the development of restenosis after stent placement has been investigated. We analyzed the association of serum lipids with the development of restenosis (≥50% diameter stenosis) of 750 lesions in 671 consecutive patients. They underwent successful stent implantation between March 93 and March 96 and had repeat angiography after a median of 191 days. For quantitative analysis an automated and computer assisted edge detection method was used. Serum levels of total cholesterol, LDL, HDL and Lp(a) were obtained at the time of the intervention and follow-up angiogram. There was no significant correlation of late lumen loss (mean: 1.1 ± 0.03 mm) within the stented segments or % restenosis (28.9%) and the levels of total cholesterol, LDL, HDL and Lp(a), neither at the time of the intervention nor at repeat angiography.

921-114 Reduction of Plasma Lipid Peroxides during low Density Lipoprotein Immunapheresis

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Extracorporal low density lipoprotein (LDL) elimination is frequently used today for the treatment of drug-resistant hypercholesterolaemic patients. One of the most specific apheresis methods, LDL-immunapheresis, was developed to selectively remove LDL and lipoprotein (a) [Lp(a)] from plasma. Since lipid peroxidation is one of the unwanted side effects of extracorporeal plasma treatments, we followed the oxidative status of patients treated with LDL-immunapheresis. For this purpose lipid hydroperoxides, the primary products of lipid peroxidation and thiobarbituric acid-reacting substances (TBARS), secondary products, were determined in 13 patients before, during and after LDL-immunapheresis. The amount of plasma volume treated per patient was 6164 ± 546 ml. Treatment led to a reduction of total cholesterol by 69 ± 8%, of LDL-cholesterol by 78.6 ± 7.3%, of HDL-cholesterol by 19.2 ± 7.1% and of triglycerides by 37.6 ± 21%. Apolipoprotein-B, Lp(a) and apoA1 were reduced by 77.1 ± 6.45%, 25 ± 5.6% and 76.4 ± 10.3%, respectively. Before treatment, mean plasma concentration of lipid hydroperoxides was 22.6 ± 15.2 μmol/L, whereas following treatment plasma hydroperoxides were reduced to 6.9 ± 7.4 μmol/L. This reduction of 64.7 ± 27% was statistically highly significant ($p < 0.001$). A non-significant change of TBARS concentration from 0.30 ± 0.26 μmol/L to 0.23 ± 0.13 μmol/L was also observed. We conclude that LDL-immunapheresis is not only very effective in reducing LDL- and Lp(a)-cholesterol, but also has a beneficial effect on the oxidative status of the patients.

921-115 Altering Physician Behavior in Lipid Testing and Therapy: Results of a Housestaff Education Program

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Treatment of hypercholesterolemia for secondary prevention of cardiac events is now firmly established. Lipid levels remain valid for 24 hours after the onset of myocardial infarction (MI) and 1994 national guidelines recommend measurement of serum lipids in patients (pts) within 24 hours in order to identify those in need of therapy. We designed a housestaff education project to alter physician behavior in obtaining lipid levels and treating elevated LDL-cholesterol in pts with acute coronary syndromes. The intervention consisted of formal didactic sessions on lipid testing and twice-weekly reinforcement sessions on rounds by the medical chief resident, along with posted reminders in clinical areas. All pts with MI during the two month study period (12/95–1/96) were identified by an elevated creatine kinase level and elevated MB fraction. Lipid testing and therapy rates were compared to a historical control population (n = 280) admitted with acute coronary syndromes over a previous recent 12 month period. *Results:* We identified 134 pts with MI. In pts for whom data was available, 35% had a history of hyperlipidemia, of whom 48% were on therapy at admission. Seventy percent of study pts compared to 45% of controls (p = 0.0001) had lipid levels drawn at some point during admission; 25% vs. 15% (p = 0.03) had fasting panels by hospital